

# Myriads of Arrhythmias and Cardiac Arrest as Modes of Presentation in Takotsubo Cardiomyopathy

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## Abstract

Takotsubo cardiomyopathy (TC) is a relative novel condition that has been increasingly reported. Studies have shown the incidence on TC to be 1-2 % of all patients presenting with acute coronary syndrome which amounts a large subset of patients. Various arrhythmias have been reported with TC, varying from benign QTc prolongation to serious life threatening ventricular arrhythmias. We present two cases of TC with myriad of ventricular arrhythmias.

with coagulopathy and upper gastrointestinal bleed. When amiodarone was omitted because of deranged liver function and prolonged QTc interval, she developed ventricular bigemini again. This was managed by giving intravenous Lignocaine and increasing the dose of oral metoprolol. Her condition stabilized after supportive management and she was discharged. On 1 month follow-up

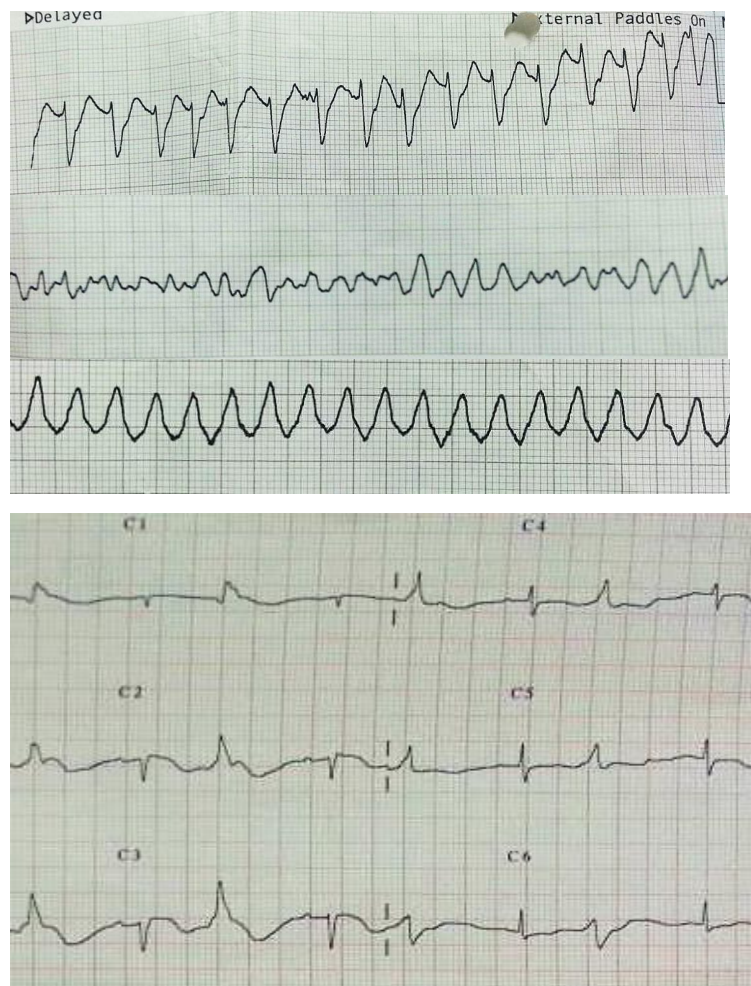
## Case Report 1

An eighty year old diabetic epileptic female patient presented with breakthrough seizures, controlled with a combination of Lorazepam and Levetiracetam. In the hospital, she had a cardiac arrest due to a polymorphic ventricular tachycardia, which was cardioverted with a 200J biphasic DC shock. Within minutes, she developed a monomorphic ventricular tachycardia which was also cardioverted with a 200J DC shock. This was followed by ventricular flutter requiring cardioversion again. Her rhythm then converted to Ventricular bigemini.

Her serial ECG showed a ST elevation in leads V1 to V3. She had several episodes of monomorphic ventricular tachycardia requiring 200J DC Shock. Her 2D Echocardiography showed akinesia of the distal interventricular septum, apex and anterolateral wall with an ejection fraction of 25%. Her cardiac enzymes were elevated but in view of the recurrent cardioversions, their value was unreliable. She was started on aspirin, ticagrelor, metoprolol and intravenous amiodarone.

She was taken up for coronary angiography in view of ST-Elevation myocardial infarction with regional wall motion abnormality on the echocardiogram and recurrent ventricular tachyarrhythmia's. Her coronary angiography showed normal vessels without any significant plaques.

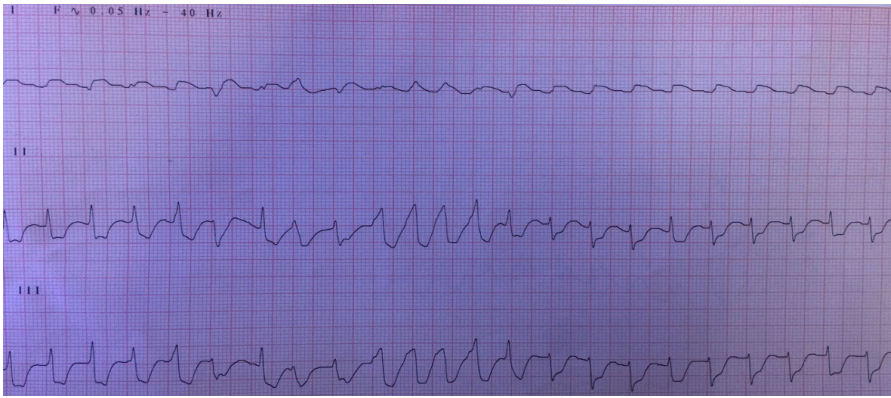
Using Mayo clinic criteria,<sup>1</sup> a probable diagnosis of *Takotsubo cardiomyopathy* was made. She was continued on metoprolol and amiodarone. She also developed ischemic hepatopathy



**Fig. 1: First Panel: Sustained Monomorphic Ventricular Tachycardia. Second Panel: Polymorphic Ventricular Tachycardia. Third Panel: Ventricular Flutter. Fourth Panel: Ventricular Bigemini. The alternate sinus beats with narrow QRS complexes show QS pattern with ST Elevation and T Inversions in Chest leads 1 to 3.**

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**Fig. 2: Polymorphic Ventricular tachycardia**

echocardiography showed a resolution of left ventricular function to normal.

## Case 2

A 60 years old lady with Multisystem Atrophy was admitted with Urinary Tract Infection. Second day of admission, she developed sudden breathlessness, for which she had to be intubated and mechanically ventilated. She was transferred to ICU where ECG showed ST Elevation in anterior leads. Her bedside Echocardiography showed Akinesia of distal Inter-ventricular septum, apex and distal antero-lateral wall of left ventricle. Her cardiac Enzymes were elevated. She was started on inotropic for hypotension.

Her Coronary Angiography revealed normal coronaries. On the basis of Mayo Clinic Criteria, probable diagnosis of Takotsubo Cardio-myopathy was made.

She persistently had high grade fever (105 degree F), her Inotropic support was gradually increasing. We decided to insert a balloon pump for hemodynamic support, especially since the use of high doses of sympathomimetic drugs could be harmful in stress cardiomyopathy. Just before the insertion of Intra-aortic balloon pump, patient developed polymorphic ventricular tachycardia (Figure 2).

Patient was given multiple DC shocks and various drugs to control (Xylocaine, Metoprolol, Diltiazem, Magnesium) the polymorphic ventricular tachycardia, but patient did not respond to any of them. Her metabolic profile showed decreased ionized calcium, which was corrected. Despite all attempts,

she persistently had polymorphic ventricular tachycardia. She developed cardiac arrest and could not be revived.

## Discussion

Takotsubo Cardiomyopathy (TC) is a relatively novel condition that has been increasingly reported. According to a study the incidence of TC is reported to be between 1 to 2 % of all acute coronary syndrome hospitalizations. The most common clinical presentations are chest pain and dyspnoea, reported in 67.8 and 17.8% of the patients, respectively. Various arrhythmias which have been reported with TC which vary from prolonged QTc to life threatening Ventricular arrhythmias. The incidence of various arrhythmias is approximately 10 %. Out of them approximately 1-3 % present with Ventricular arrhythmias and around 3 % of patients with Sudden cardiac death.<sup>2</sup>

A large registry based data which included a total of 6837 patients, majority of patients (78%) had no documented arrhythmias. Remaining cases had atrial arrhythmias as the predominant arrhythmias; 11% (752) of them had atrial fibrillation, 5% (342) had paroxysmal supraventricular tachycardia and 1% (68) of them had atrial flutter. Ventricular arrhythmias were present in only 2% (137) of patients while 3% (205) presented as sudden cardiac death.<sup>3</sup>

The etiopathogenesis of TC is not well understood. Whether TC is cause or effect of arrhythmias remains a question unanswered. A

study by Wittstein et al. showed that plasma catecholamine levels in TC at presentation are raised above and beyond those resulting from MI and left ventricular failure (in comparison with Killip class III MI).<sup>4</sup> Cardiac MRI based studies have given an understanding of the evolution of TC at a tissue level, characteristically with an absence of scar formation. This makes re-entry an unlikely mechanism for arrhythmia and lends support to catecholaminergic, automatic, or fascicular tachycardias as more likely entities, with related cytosolic calcium overload.<sup>5</sup> Coronary artery spasm has been reported as a cause of TC, and in these circumstances, there may be a transient ischemic trigger for arrhythmias.<sup>6</sup>

## Conclusion

Takotsubo cardiomyopathy is being diagnosed more frequently and arrhythmias can be a part of the clinical presentation. The arrhythmias may range from benign atrial arrhythmias to life threatening ventricular arrhythmias and even sudden cardiac death. We encountered two patients with TC who had a cardiac arrest due to ventricular arrhythmias. Although one of them responded to medical management and recovered, the other succumbed to the incessant ventricular arrhythmia. It is important to recognize these arrhythmias as a part of the clinical scenario of Takotsubo cardiomyopathy.

## References

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